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# ECONOMIC GROWTH, POPULATION THEORY, AND PHYSIOLOGY: THE BEARING OF LONG-TERM PROCESSES ON THE MAKING OF ECONOMIC POLICY

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Prepared for presentation as the Prize Lecture in Economic Sciences in Memory of Alfred Nobel, December 9, 1993. Since this lecture is based on research still in progress, it is important to emphasize that the findings reported here are provisional and are subject to change as the current databases expand and as the analyses of these data are refined. Nevertheless, I believe that the general outlines of the emerging new theories of mortality, morbidity, and aging are likely to survive further research. Research reported in this paper was supported by grants from the National Institutes of Health (#PO1-AG10120-02), the National Science Foundation, (#SES-9114981), and the Walgreen Foundation. I have benefitted from comments and criticisms by Christopher J. Acito, Robert McC. Adams, Gary S. Becker, Christine K. Cassel, Katherine A. Chavigny, Dora L. Costa, William J. Darby, Partha Dasgupta, Sidney Davidson, Stanley L. Engerman, Phyllis Eveleth; Enid M. Fogel, Milton Friedman, Victor R. Fuchs, Zvi Griliches, Robin M. Hogarth, Susan E. Jones, John M. Kim, Peter Laslett, Lionel W. McKenzie, Reynaldo Martorell, Douglass C. North, S. Jay Olshansky, Clayne L. Pope, Samuel H. Preston, Irwin Rosenberg, Roger A. Schofield, Nevin S. Scrimshaw, Robert M. Solow, Richard H. Steckel, David Surdam, Richard Suzman, James M. Tanner, Peter Temin, James Trussell, James W. Vaupel, Hans T. Waaler, and E.A. Wrigley. This paper is part of NBER's research programs in Growth, Health Economics, and the Development of the American Economy. Any opinions expressed are those of the author and not those of the National Bureau of Economic Research.

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## ABSTRACT

This paper sketches a theory of the secular decline in morbidity and mortality that takes account of changes in human physiology since 1700. The synergism between technological and physiological improvements has produced a form of human evolution, much more rapid than natural selection, which is still ongoing in both OECD and developing countries. Thermodynamic and physiological aspects of economic growth are defined and their impact on growth rates is assessed. Implications of this theory for population forecasting, measurement of national income, demand for leisure, pension policies, and for the demand for health care are considered.

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## Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the Making of Economic Policy

Economic history has contributed significantly to the formulation of economic theory. Among the economists who have found history an important source for their ideas are Smith, Malthus, Marx, Marshall, Keynes, Hicks, Arrow, Friedman, Solow, and Becker. Failure to take account of history, as Simon Kuznets (1941) stressed, has often led to a misunderstanding of current economic problems by investigators who have not realized that their generalizations rested upon transient circumstances. Nowhere is the need to recognize the role of long-run dynamics more relevant than in such pressing current issues as medical care, pension policies, and development policies. I will attempt to put these issues into perspective by describing the escape from hunger and premature mortality which began in Europe and North America about 300 years ago and has not yet run its course. Even in the rich countries of the world, most individuals are still developing chronic diseases and dying prematurely.

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The attempt to explain the secular decline in mortality in a systematic way did not begin until after World War I because before that time it was uncertain whether such a decline was in progress. There were two reasons for the delay in recognizing the phenomenon. First, little was known about mortality rates before the end of the Napoleonic wars. Hardly a dozen life tables had been constructed before 1815 by various pioneers in demography and they exhibited no clear time trend (Dublin and Lotka 1936; Gille 1949/50). Second, there was little evidence in the first four official English life tables, covering the years 1831-80, of a downward trend in mortality.

By the third decade of the twentieth century, however, it became obvious that the new declines in British mortality rates were not just a cyclical phenomenon. Between 1871 and 1901 life expectation in Britain increased by 4 years. During the next three decades there was an additional gain of 16 years. Similar declines in mortality were recorded in other European nations.

The plunge in mortality rates during the early decades of the twentieth century delivered a major blow to the Malthusian theory of population. Improvements in mortality were supposed to be short lived because, under the conditions of population pressure against the food supply that Malthus specified, the elimination of deaths due to one disease would be replaced by those due to some other malady. Efforts to reconcile Malthusian doctrine with the observed mortality decline, to modify it, or to replace it produced a large new literature.

### *1. Explaining the Secular Decline in Mortality*

The drive to explain the secular decline in mortality pushed research in three directions. First, there was a concerted effort to develop time series of death rates that extended as far back in time as possible in order to determine just when the decline in mortality began. Second, the available data on mortality rates were analyzed in order to identify factors that might explain the decline as well as to establish patterns or "laws" that would allow predictions of the future course of mortality.

Third, a widespread effort was undertaken to determine the relationship between the food supply and mortality rates. There were several aspects to this effort. Perhaps the most important was the emergence of a science of nutrition that identified a series of diseases related to specific nutritional deficiencies and discovered the synergy between nutrition and infection (Scrimshaw, Taylor and Gordon 1968). Another aspect was the emergence of the field of development economics after World War II as part of the campaign to close the yawning gap in income, health, and life expectancy between the industrialized nations and the "developing nations." Still another aspect was the combined effort of economic and demographic historians to study the role of mortality crises and their relationship to famines during the seventeenth and eighteenth centuries.

Prior to the 1960s, efforts to reconstruct the secular trend in European mortality were focused primarily on notable local communities and parishes. However, developments in

statistical techniques and the remarkable reductions in computational costs during the 1960s and 1970s made it possible to draw and process large nationally representative samples. The results of these efforts, combined with official statistics after 1830 in France and after 1871 in England, are displayed in Figure 1. Analysis of the French and English series revealed that the secular decline in mortality took place in two waves. In the English case the first wave began during the second quarter of the eighteenth century (18-II) and lasted through 19-I after which mortality rates stabilized for half a century. The decline resumed during 19-IV and continues through the present. The French case is similar except that the first wave of the decline in mortality began about half a century earlier in France and its rate of decline during the first wave was more rapid.

Perhaps the most surprising aspect of Figure 1 is the implication that the elimination of crisis mortality, whether related to famines or not, accounted for less than 10 percent of the secular decline in mortality rates (Wrigley and Schofield 1981; Lee 1981; Dupâquier 1989; Weir 1982 and 1989; Richards 1984; Galloway 1986; Fogel 1992b). Similar results were obtained by studies of official statistics for Sweden (Bengtsson and Ohlsson 1984, 1985; Galloway 1987; cf. Eckstein, Schultz, and Wolpin 1985; Perrenoud 1984 and 1991; Fridlizius 1984). By demonstrating that famines and famine mortality are a secondary issue in the escape from the high mortality rates of the early modern era, these studies shifted attention to the neglected issue of chronic malnutrition as the principal pathway through which malnutrition contributed to the high mortality rates of the past (cf. Sen 1981).

## *2. The Synergy Between Biomedical and Economic Analyses of the Secular Trend in Chronic Malnutrition*

Recently developed biomedical techniques, when integrated with economic techniques, make it possible to probe deeply into the extent of chronic malnutrition from the beginning of the eighteenth century in Europe and North America, to chart and explain the escape from such malnutrition, and to consider the impact of improved nutrition on the secular trend in health and

life expectation, on labor productivity, and on economic growth. The combination of the economic and biomedical modes of analysis has been synergistic since it has yielded analytical insights that could not have been obtained merely by relying on the techniques of one of the disciplines.

Malnutrition can be caused either by an inadequate diet or by claims on that diet (including work and disease) so great as to produce malnutrition despite a nutrient intake that in other circumstances might be deemed adequate.[1] There can be little doubt that the high disease rates prevalent during the early modern era would have caused malnutrition even with diets otherwise adequate in calories, protein, and other critical nutrients. However, recent research indicates that, for many European nations before the middle of the nineteenth century, the national production of food was at such low levels that the poorer classes were bound to have been malnourished under any conceivable circumstance, and that the high disease rates of the period were not merely a cause of malnutrition but undoubtedly, to a considerable degree, a consequence of exceedingly poor diets.

*2.1 Energy Cost Accounting and Secular Trends in Body Size*

As a result of the work of agricultural historians we now have estimates of British agricultural production by half-century intervals going back to 1700. These provide the basis for national food balance sheets which indicate the secular trend in British caloric consumption (Chartres 1985; Holderness 1989; Allen 1994). Supplemented by household surveys of food purchases (Shammas 1984 and 1990; Oddy 1990; cf. Fogel 1987), these sources indicate that average daily caloric consumption in Britain c.1790 was about 2,060 kcal per capita or about 2,700 kcal per consuming unit (equivalent adult males). For France, Toutain (1971) has constructed estimates from national food balance sheets going back to the decade preceding the French Revolution. His estimates indicate that the daily per capita caloric consumption was 1,753 kcal during 1781-90 and 1,846 during 1803-12. Converted into calories per consuming

unit, these figures become 2,290 and 2,410.

One implication of these estimates is that mature adults of the late eighteenth century must have been very small by current standards. Today the typical American male in his early thirties is about 177 cm (69.7 inches) tall and weighs about 78 kg (172 lbs) (USDHHS 1987). Such a male requires daily about 1,794 kcal for basal metabolism (the energy required to keep the body functioning while at rest) and a total of 2,279 kcal for baseline maintenance (the 1,794 kcal required for basal metabolism plus 485 kcal for digestion of food and vital hygiene) (Quenouille et. al. 1951; FAO/UNU/WHO 1985). If either the British or the French had been that large during the eighteenth century, virtually all of the energy produced by their food supplies would have been required for maintenance and hardly any would have been available to sustain work. To have the energy necessary to produce the national products of these two countries c.1700, the typical adult male must have been quite short and very light.

This inference is supported by data on stature and weight which have been collected for European nations. Table 1 provides estimates of final heights of adult males who reached maturity between 1750 and 1875. It shows that during the eighteenth and nineteenth centuries Europeans were severely stunted by modern standards (cf. line 6 of Table 1). Estimates of weights for European nations before 1860 are much more patchy. Those which are available, mostly inferential, suggest that c.1790 the average weight of English males in their thirties was about 61 kg (134 lbs), which is about 20 percent below current levels. The corresponding figure for French males c.1790 may have been only about 50 kg (about 110 lbs), which is about a third below current standards.

*2.2 Size Distributions of Calories*

The synergy between the economic and biomedical lines of analysis is apparent in the new insights obtained by switching from a reliance on the mean height, the mean weights, and the mean daily consumption of nutrients to the size distributions of these variables. Because of

the limits of time, I focus here on the distributions of calories.[2]

Size distributions of caloric consumption are one of the most potent instruments in assessing the plausibility of proffered estimates of average diets. They not only bear on the implications of a given level of caloric consumption for morbidity and mortality rates, but they also indicate whether the calories available for work are consistent with the level of agricultural output and with the distribution of the labor force between agriculture and nonagriculture (Fogel and Floud 1994; Fogel 1991; cf. Wrigley 1987). Although national food-balance sheets, such as those constructed by Toutain (1971) for France over the period 1781-1952, provide mean values of per capita caloric consumption, they do not produce estimates of the size distribution of calories.[3]

Three factors make it possible to estimate the size distributions of calories from the patchy evidence available to historians. First, studies covering a wide range of countries indicate that distributions of calories are well described by the lognormal distribution. Second, the variation in the distribution of calories (as measured by the coefficient of variation [$s/\bar{X}$] or the Gini [$G$] ratio) is far more limited than the distribution of income. Third, when the mean of the distribution is known, the coefficient of variation (which together with the mean determines the distribution), can be estimated from information in either tail of the distribution. Fortunately, even in places and periods where little is known about ordinary people, there is a relative abundance of information about the rich. At the bottom end, it is demographic information, particularly the death rate, which rather tightly constrains the proportion of the population whose average daily consumption of calories could have been below BMR or baseline maintenance.

Table 2 shows the exceedingly low level of work capacity permitted by the food supply in France and England c.1790, even after allowing for the reduced requirements for maintenance because of small stature and body mass (cf. Freudenberger and Cummins 1976). In France the bottom 10 percent of the labor force lacked the energy for regular work and the next 10 percent had enough energy for less than 3 hours of light work daily (0.52 hours of heavy work).

Although the English situation was somewhat better, the bottom 3 percent of its labor force lacked the energy for any work, but the balance of the bottom 20 percent had enough energy for about 6 hours of light work (1.09 hours of heavy work) each day.

Table 2 also points up the problem with the assumption that for *ancien régime* populations, a caloric intake that averaged 2,000 kcal per capita (2,600 per consuming unit) daily was adequate (Livi-Bacci 1990). That average level of consumption falls between the levels experienced by the French and the English c.1790. In populations experiencing such low levels of average consumption, the bottom 20 percent subsisted on such poor diets that they were effectively excluded from the labor force with many of them lacking the energy even for a few hours of strolling. That appears to be the principal factor explaining why beggars constituted as much as a fifth of the populations of *ancien régimes* (Goubert 1973; Cipolla 1980; Laslett 1984). Even the majority of those in the top 40 percent of the caloric distribution were so stunted (height below U.S. standards) and wasted (weight below U.S. standards) that they were at substantially higher risk of incurring chronic health conditions and of premature mortality (see the next section).

### *3. Waaler Curves and Surfaces: A New Analytical Tool*

Extensive clinical and epidemiological studies over the past two decades have shown that height at given ages, weight at given ages, and weight-for-height (a body mass index, or BMI) are effective predictors of the risk of morbidity and mortality. Until recently most of the studies have focused on children under age 5, using one or more of the anthropometric indicators at these ages to assess risks of morbidity and mortality in early childhood, and it was at these ages that the relevance of anthropometric measures originally were established most firmly (Sommer and Lowenstein 1975; Chen, Chowdhury, and Huffman 1980; Billewicz and MacGregor 1982; Kielmann et al. 1983; Martorell 1985). During the last few years, however, a considerable body of evidence has accumulated suggesting that height at maturity is also an important predictor of

the probability of dying and of developing chronic diseases at middle and late ages (Marmot, Shipley, and Rose 1984; Waaler 1984; John 1988; Costa 1993; Kim 1993). BMI has similar predictive properties (Heywood 1983; Waaler 1984; Martorell 1985; Payne 1992; Osmani 1992; cf. Srinivasan 1992).

Height and BMI measure different aspects of malnutrition and health. Height is a net rather than a gross measure of nutrition. Moreover, although changes in height during the growing years are sensitive to current levels of nutrition, mean final height reflects the accumulated past nutritional experience of individuals throughout their growing years, including the fetal period. It follows that when final heights are used to explain differences in adult mortality rates, they reveal the effect, not of adult levels of nutrition on adult mortality rates, but of nutritional levels during infancy, childhood, and adolescence on adult mortality rates. A weight-for-height index, on the other hand, reflects primarily the current nutritional status. It is also a net measure in the sense that BMI reflects the balance between current intakes and the claims on those intakes.

*3.1 The Relationship Between Body Size and the Risk of Death at Middle and Late Ages*

A number of recent studies have established the predictive power of height and BMI with respect to morbidity and mortality at later ages. The results of two of these studies are summarized in Figures 2 and 3. Part A of Figure 2 reproduces a diagram by Waaler (1984). It shows that short Norwegian men aged 40-59 at risk between 1963 and 1979 were much more likely to die than tall men. Indeed, the risk of mortality for men with heights of 165 cm (65.0 inches) was on average 71 percent greater than that of men who measure 182.5 cm (71.9 inches). Part B shows that height is also an important predictor of the relative likelihood that men aged 23-49 would be rejected from the Union Army during 1861-65 because of chronic diseases. Despite significant differences in ethnicities, environmental circumstances, the array and severity of diseases, and time, the functional relationship between height and relative risk

are strikingly similar in the two cases.

Waaler (1984) has also studied the relationship in Norway between BMI and the risk of death in a sample of 1.7 million individuals. Curves summarizing his findings are shown in Figure 3 for both men and women. Although the observed values of the BMI ($kg/m^2$) ranged between 17 and 39, over 80 percent of the males over age 40 had BMI's within the range 21-29. Within the range 22-28, the curve is relatively flat, with the relative risk of mortality hovering close to 1.0. However, at BMIs of less than 22 and over 28, the risk of death rises quite sharply as the BMI moves away from its mean value. It will be noticed that the BMI curves are much more symmetrical than the height curves in Figure 2, which indicates that high BMIs are as risky as low ones.

Although Figures 2 and 3 are revealing, neither one singly, nor both together, are sufficient to shed light on the debate over whether moderate stunting impairs health when weight-for-height is adequate, since Figure 2 is not controlled for weight and Figure 3 is only partially controlled for height (Fogel 1987; Fogel and Floud 1994). To get at the "small-but-healthy" issue one needs an iso-mortality surface that relates the risk of death to both height and weight simultaneously. Such a surface, presented in Figure 4, was fitted to Waaler's data by a procedure described elsewhere (Fogel 1993b). Transecting the iso-mortality map are lines which give the locus of BMI between 16 and 34, and a curve giving the weights that minimize risk at each height.

Figure 4 shows that even when body weight is maintained at what Figure 3 indicates is an "ideal" level (BMI = 25), short men are at substantially greater risk of death than tall men. Thus, an adult male with a BMI of 25 who is 164 cm tall is at about 55 percent greater risk of death than a male at 183 cm who also has a BMI of 25. Figure 4 also shows that the "ideal" BMI (the BMI that minimizes the risk of death) varies with height. A BMI of 25 is "ideal" for men in the neighborhood of 176 cm, but for tall men (greater than 183 cm) the ideal BMI is

between 22 and 24, while for short men (under 168 cm) the "ideal" BMI is about 26.

*3.2 Using Waaler Surfaces to Explain the Secular Decline in Mortality*

Superimposed on Figure 4 are rough estimates of heights and weights in France at 4 dates. In 1705 the per capita food supply in France was lower than in Britain so that average body mass was probably even lower than in Britain. Circa 1705 the French probably achieved equilibrium with their food supply at an average height of about 161 cm and BMI of about 18. Over the next 270 years the food supply expanded with sufficient rapidity to permit both the height and the weight of adult males to increase. Figure 4 indicates that it was factors associated with the gain in BMI that accounted for most of the reduction in the risk of mortality before 1870. After 1870, factors associated with the gain in height explain most of the additional mortality decline. Figure 4 also implies that while these factors jointly explain about 90 percent of the decline in French mortality rates over the period between c.1785 and c.1870, they only explain about 50 percent of the decline in mortality rates during the past century. Increases in body size and the factors associated with it continued to have a major impact on the gains in life expectation among persons of relatively good nutritional status, but during the last century factors other than those related to height and BMI became increasingly important.

The analysis in this section points to the misleading nature of the concept of subsistence as Malthus originally used it and as it is still widely used today. Subsistence is not located at the edge of a nutritional cliff, beyond which lies demographic disaster. The evidence outlined in the paper implies that rather than one level of subsistence, there are numerous levels at which a population and a food supply can be in equilibrium, in the sense that they can be indefinitely sustained. However, some levels will have smaller people and higher "normal" (non-crisis) mortality than others.[4]

*3.3 The Relevance of Waaler Surfaces for Predicting Trends in Chronic Diseases*

Poor body builds increased vulnerability to diseases, not just contagious diseases, but

chronic diseases as well. This point is implicit in Figure 2 which shows that chronic conditions were much more frequent among short young men in the 1860s than among tall men. Figure 5 shows that the same relationship between ill health and stature exists among the males covered by the U.S. National Health Interview Surveys (NHIS) for 1985-88. Stunting during developmental ages had a long reach and increased the likelihood that people would suffer from chronic diseases at middle and at late ages.

American males born during the second quarter of the nineteenth century were not only stunted by today's standards, but their BMIs at adult ages were about 15 percent lower than current U.S. levels (Fogel, Costa, and Kim 1993). The implication of the combined stunting and low BMI is brought out by Figure 6 which presents a Waaler surface for morbidity estimated by Kim (1993) from NHIS data for 1985-88.

The Waaler surface for risk from chronic conditions in Figure 6 is similar to, but not identical with, the Norwegian surface for mortality (see Figure 4). The iso-morbidity curves in ill health rise more steeply than the iso-mortality curves as one moves away in either direction from the optimal weight curve. Furthermore, the optimal weight curve in Figure 6 usually lies about one iso-BMI curve to the right of the optimal weight curve computed from the Norwegian mortality data. Thus, both the Norwegian mortality data and the U.S. health data indicate that for men in the neighborhood of 1.60-1.65 meters the optimal BMI is in the range of 25 to 27. This is above current levels recommended by FAO/WHO/UNU (1985), falling into the lower ranges of overweight in that standard.

Figure 6 also presents the coordinates in height and BMI of Union Army veterans who were 65 or over in 1910 and of veterans (mainly of World War II) who were the same ages during 1985-88. These coordinates predict a decline of about 35 percent in the prevalence of chronic disease among the two cohorts. About 61 percent of the predicted decline in ill health is due to factors associated with the increase in BMI and the balance is due to factors associated

with increased stature.

The decline in the prevalence of chronic diseases predicted by Figure 6 is quite close to what actually occurred. Table 3 compares the prevalence of chronic diseases among Union Army men aged 65 and over in 1910 with two surveys of veterans of the same ages in the 1980s. That table indicates that heart disease was 2.9 times as prevalent, musculoskeletal and respiratory diseases were 1.6 times as prevalent, and digestive diseases were 4.7 times as prevalent among veterans aged 65 or over in 1910 as in 1985-88. During the 7.6 decades separating the two groups, the prevalence of heart disease among the elderly declined at a rate of 12.8 percent per decade, while musculoskeletal and respiratory diseases each declined at a rate of 5.9 percent per decade.

Young adults born between 1822 and 1845 who survived the deadly infectious diseases of childhood and adolescence were not freer of degenerative diseases than persons of the same ages today, as some have suggested, but more afflicted. At ages 35-39 hernia rates, for example, were more than three times as prevalent in the 1860s as in the 1980s. Of special note is the much higher incidence of clubfoot in the 1860s -- a birth anomaly which suggests that the uterus was far less safe for those awaiting birth than it is today.

Those who also survived diseases of middle ages were more afflicted by degenerative chronic conditions at old ages in the 1910s than in the 1980s. Nearly 74 percent of the elderly Union Army veterans suffered from three or more disabling chronic conditions, which is much higher than the rate among elderly veterans in 1983 (Fogel, Costa, and Kim 1993). It may be true that there were less genetically frail persons among those who survived to age 65 in 1910 than there are today. If so, that genetic advantage was apparently offset by a lifetime of socioeconomic and biomedical stress that left health in old age badly impaired and that sharply curtailed the life expectations of the elderly. During the 1910s the elderly died not from the infectious diseases that killed the great majority of their cohorts at relatively young ages but

primarily from degenerative diseases which, at the two-digit level of the International Classification of Diseases, are similar to the distribution of causes of death during the 1980s, except that deaths from neoplasms were lower and deaths from tuberculosis were higher than in the 1980s.

The provisional findings thus suggest that chronic conditions were far more prevalent throughout the life-cycle for those who reached age 65 before World War I than is suggested by the theory of the epidemiological transition. Reliance on causes-of-death information to characterize the epidemiology of the past has led to a significant misrepresentation of the distribution of health conditions among the living. It has also promoted the view that the epidemiology of chronic diseases is more separate from that of contagious diseases than now appears to be the case.

### *4. Physiological Foundations for Waaler Surfaces and Curves*

What is the basis for the predictive capacity of Waaler surfaces and curves? Part of the answer resides in the realm of human physiology, which concerns the functioning of the organs and the organ systems of the body. Variations in height and weight appear to be associated with variations in the chemical composition of the tissues that make up these organs, in the quality of the electrical transmission across membranes, and in the functioning of the endocrine system and other vital systems.

Research in this area is developing rapidly and some of the new findings are yet to be confirmed. The exact mechanisms by which malnutrition and trauma in utero or early childhood are transformed into organ dysfunctions are still unclear. What is agreed upon is that the basic structure of most organs are laid down early, and it is reasonable to infer that poorly developed organs may break down earlier than well developed ones. The principal evidence so far is statistical and, despite agreement on certain specific dysfunctions, there is no generally accepted theory of cellular aging (cf. Tanner 1990 and 1993).

With these caveats in mind, recent research bearing on the connection between malnutrition and body size and the later onset of chronic diseases can conveniently be divided into three categories. The first category involves forms of malnutrition (including the ingestion of toxic substances) that cause permanent, promptly visible physiological damage, as is seen in the impairment of the nervous systems of fetuses due to excess consumption of alcohol or of smoking by pregnant women. Alcohol, for example, induces growth retardation in fetuses and infants and causes atrial septal defect, microcephaly, and other birth anomalies which are collectively labeled Fetal Alcohol Syndrome or Fetal Alcohol Symptoms (Robbins, Cotran, Kumar 1984). It appears that protein calorie malnutrition (PCM) in infancy and early childhood can lead to a permanent impairment of central nervous system function (Scrimshaw and Gordon 1968; Martorell, Rivera, and Kaplowitz 1990; Chavez, Martinez, and Soberanes 1993; cf. Volpe 1987). Iodine deficiency in utero and moderate-to-severe iron deficiency during infancy also appear to cause permanent neurological damage (Lozoff, Jimenez, and Wolf 1991; Scrimshaw 1993).

Not all damage due to retarded development in utero or infancy caused by malnutrition shows up immediately. In a recent series of studies D.J.P. Barker and his colleagues (Barker et al. 1989; Barker, Osmond, and Golding 1990; Barker 1991; Barker et al. 1992; Law et al. 1993; Phillips et al. 1993; Phipps et al. 1993) have reported that such conditions as coronary heart disease, hypertension, stroke, diabetes, and autoimmune thyroiditis begin in utero or in infancy, but do not become apparent until midadult or later ages. In these cases, individuals appear to be in good health, and function well in the interim. However, early onset of the degenerative diseases of old age appears to be linked to inadequate cellular development early in life. Some, but not all, such cases are associated with low birth weight. Some babies are born in the normal weight range but experience below average infant weight gains. In other instances babies are small relative to the size of their placentas, short in relation to the size of

their head, or long but thin (Barker 1993; cf. Tanner 1993).

Certain physiological dysfunctions incurred by persons suffering from malnutrition can, in principle, be reversed by improved dietary intake, but they often persist because the cause of the malnutrition persists. If the malnutrition persists long enough these conditions can become irreversible or fatal. This category of dysfunctions includes the degradation of tissue structure, especially in such vital organs as the lungs, the heart, and the gastrointestinal tract. In the case of the respiratory system, for example, there is not only decreased muscle mass and strength but also impaired ventilatory drive, biochemical changes in connective tissue, and electrolyte abnormalities. Malnutrition also has been related to the atrophy of the mucosal cells of the gut, the inhibition of wound healing, increased likelihood of traumatic shock and of sepsis, impaired functioning of the endocrine system, increased tendency to edema, electrical instability that can provoke acute arrhythmias, and degenerative joint diseases (Saba, Dillon, and Lanser 1983; Idiaquez 1988; McMahon and Bistrian 1990; Hill 1990; Fisler 1992; cf. Manton 1993).

Also relevant is the discovery of the relationship between birth weight and the probability of neonatal death. The curves in Figure 7 are "U" shaped, indicating that in each population babies significantly heavier than the optimal weight also incur high mortality risks. Moreover the optimal birth weight in the two populations with small mothers was significantly lower than that of the U.S. population where mothers were relatively large. It appears that prior to high-technology interventions, the size of the mother's pelvis constrained the rate at which birth size (and perhaps the robustness of the vital organs of the baby) could have increased with the improvement in intrauterine nutrition. Babies at weights which optimized survival in a relatively tall population were at elevated risks of dying in populations with relatively short mothers, due to delivery distress. There was, in other words, an intergenerational constraint on the rate at which babies could escape from the effects of malnutrition as fetal nutrition improved (cf. Chandra 1975).

The recent physiological findings also cast new light on the first phase of the secular decline in mortality. Some investigators have called attention to changes in age-specific mortality rates that may indicate a shift in the balance between pathogens and their human hosts (Fridlizius 1984; Perrenoud 1984). Although the shift has been attributed to a decline in the virulence of pathogens, not enough evidence is in hand as yet to assess this possibility. However, the recent physiological research summarized in this section suggests a new pathway through which the balance between pathogens and human hosts may have turned in favor of the hosts. In addition to the improved operation of the immune system, there is the increased capacity of a vital organ to survive the attack of pathogens as a result of increased tissue resilience, including the improved operation of the nervous system. The process could have been synergistic since the improvement in the operation of the immune system might have interacted with the increased resilience of other vital organs.[5] The last possibility is consistent with the age-specific patterns of decline in mortality rates that have so far been uncovered for the eighteenth century.

### *5. Some Implications for Current Policy*

Malthus believed that malnutrition manifested itself in the exceptional--in periodic famines and in the excess mortality prevalent among the ultra-poor of his day who lived in misery and vice. He thought that persons near the middle of the social order, the sturdy agricultural laborer or the town artisan, were generally well fed, healthy, and lived normal life spans.

We now know, however, that famines accounted for less than 4 percent of the premature mortality of Malthus's age, and that the excess mortality of the ultra-poor (the bottom fifth of society) accounted for another sixth of premature mortality. About two-thirds of all premature mortality in Malthus's time came from the part of society that Malthus viewed as productive and healthy. Yet by current standards, even persons in the top half of the income distribution in

Britain during the eighteenth century were stunted and wasted, suffered far more extensively from chronic diseases at young adult and middle ages than is true today, and died 30 years sooner than today.[6]

*5.1 Implications for Poor Countries*

The Malthusian legacy is embodied in such theses as "small-but-healthy" which holds that stunted or moderately wasted individuals may not be more vulnerable to ill health and mortality than those who conform to the U.S. standard. The paucity of life-cycle data sets in developing countries caused investigators to focus only on the early years of the life span, searching for interactions between natal or infant measures of size and measures of health and work capacity later in childhood. Such studies generally picked up the effects of only exceedingly severe stunting and wasting (more than 2 standard deviations below average), missing the impact of more moderate size effects, many of which do not show up until later in life (Seckler 1980; Sukhatme 1981; Lipton 1983; cf. West et al. 1990).

However, the information reported in this paper indicates that childhood stunting and wasting has a long reach, predicting chronic disease rates at young adult and later ages. The higher prevalence of disabling chronic diseases among adults in the developing nations has escaped attention because the relevant information on such conditions are not generally collected. But the long reach of childhood malnutrition in rich countries, both now and when they were much poorer than they are today, suggests that a similar interconnection also exists in developing countries.

Chronic diseases are not the only way that chronic malnutrition reduces the productivity of the labor force. When the mean amounts of calories are as low as they are in the poor nations of the world, labor force participation rates and measures of labor productivity are bound to be low, especially when the hours of labor are adjusted for the intensity of labor (see Fogel 1991; cf. Dasgupta 1993). Elsewhere I have estimated that when the labor input is adjusted for

intensity (measured by calories), improved gross nutrition accounts for roughly 30 percent of the growth of per capita income in Britain between 1790 and 1980.

*5.2 Implications for Rich Countries*

Between 1850 and 1950 U. S. life expectation at birth increased from about 40 to 68 years. Then for the next two decades further progress in longevity came to a virtual halt. During and following this interregnum investigators who reviewed the progress in mortality over the preceding century tended toward a consensus on three propositions: (1) The century-long decline in mortality rates was unique and could not be repeated because virtually all of the gains made through the elimination of death from contagious diseases below age 60 had been made. (2) Deaths, now concentrated at older ages, were due to degenerative diseases that were unrelated to the contagious diseases that they superseded. The degenerative diseases were caused by accelerated organ losses that were part of the natural process of aging. (3) There was an upper limit to life expectation that was genetically determined. One influential paper put that limit at 85 $\pm$ 7 years (Fries 1980, cf. Fries 1989).

More recent studies, responding to the renewed decline in mortality, which this time is concentrated at ages 65 and over, have uncovered evidence that militates against the notion of a genetically fixed life span or, if it is fixed, suggests that the upper limit is well above 85. Vaupel's study of Danish twins indicates that genetic factors account for only about 30 percent of the variance in age of death (Vaupel 1991a). His study of Swedish males who lived to age 90 indicates that the death rate at that age has declined at a rate of about 1 percent per annum since 1950, a finding that is contradictory to the rectangularization of the survivorship curve (Vaupel 1991b; cf. Thatcher 1992; Vaupel and Lundstrom 1994; Kannisto et al. 1994). Two recent studies of insect populations (Carey et al. 1992; Curtsinger et al. 1992) indicated that variation in environmental conditions had a much larger effect on the life span than genetic factors and revealed no pattern suggestive of a fixed upper limit. Collectively, these studies do

not rule out genetic factors but suggest something much less rigid than the genetic programming of absolute life spans: An emerging theory combines genetic susceptibility of various organs with cumulative insults as a result of exposure to risk.

Recent studies also indicate that age-specific rates of chronic conditions above age 65 are generally falling. According to Manton, Corder, and Stallard (1993) the rate of disability among the elderly in the U. S. declined by 4.7 percent between 1982 and 1989. Put on a decade basis, this rate of decline is quite similar to the long-term rates of decline between 1910 and 1985-88 in chronic conditions among elderly veterans (Fogel, Costa, and Kim 1993). The finding is consistent with the growing body of evidence (reported in section 3 and 4 above) indicating that chronic diseases at later ages are, to a considerable degree, the result of exposure to infectious diseases, malnutrition, and other types of biomedical and socioeconomic stress early in life. It is also consistent with the predicted decline of about 6 percent per decade in chronic diseases based on the Waaler surface in ill health displayed in Figure 6 (cf. Blair et al. 1989; Manton, Stallard, and Singer 1992; Manton and Soldo 1992).

Much current research is now focused on explaining the decline in chronic conditions. Part of the emerging explanation is a change in life styles, particularly reduced smoking, improved nutrition, and increased exercise, which appear to be involved in reducing the prevalence of coronary heart disease and respiratory diseases. Another part of the explanation is the increasing effectiveness of medical intervention. This point is strikingly demonstrated by comparing the second and last columns of the line on hernias in Table 3, above. Prior to World War II hernias, once they occurred, were generally permanent and often exceedingly painful conditions. However, by the 1980s about three-quarters of all veterans who ever had hernias were cured of them. Similar progress over the seven decades is indicated by the line on genito-urinary conditions. Other areas where medical intervention has been highly effective include control of hypertension and reduction in the incidence of stroke, surgical removal of

osteoarthritis, replacement of knee and hip joints, curing of cataracts, and chemotherapies that reduce the incidence of osteoporosis and heart disease (Manton, Corder, and Stallard 1993).

The success in medical interventions combined with rising incomes has naturally led to a huge increase in the demand for medical services. Econometric estimates suggest a long-run income elasticity in the demand for medical services across OECD nations in the neighborhood of 1.5 and indicate that 90 percent of the variance in medical expenditures across OECD countries is explained by variations in income (Moore, Newman, and Fheili 1992). The rapidly growing level of demand, combined with the egalitarian policy of providing medical care at highly subsidized prices, has created the crisis in health care costs that is now such a focus of public policy debates across OECD nations, with various combinations of price and governmental rationing under consideration (Economist 1990; Newhouse 1992; Schwartz and Aaron 1991, Schieber, Poullier, and Greenwald 1993).

Whatever the eventual outcome of these policy debates, it is clear that we are in a much different world than that of Malthus. Instead of debating whether to provide food to paupers who might otherwise die, we are now debating how to distribute services that have proved successful in raising the quality of life of the aged and in extending life expectation. And we are now struggling with entirely new ethical issues such as whether it is right to restrict medical services that extend life of a low quality (Shuttleworth 1990; Wolfe 1986; Pellegrino 1993).

Growing opportunity to improve health at young ages, to reduce the incidence of chronic diseases at late ages, and to cure or alleviate the disabilities associated with chronic diseases raises two other post-Malthusian population issues. One is the impact of improved health on population size. A recent paper by Ahlburg and Vaupel (1990) pointed out that if mortality rates at older ages continue to decline at 2 percent per annum, the U.S. elderly population of 2050 would be 36 million larger than forecast by the Census Bureau (cf. Preston 1993). That possibility poses policy issues with respect to health care costs (because total medical costs may

rise sharply even if cure rates continue to improve) and to pension costs (because the number of persons eligible for benefits under present proposed rules and of projected levels of compensation will become so large that outpayments will exceed planned reserves).

Some policymakers have sought to meet the pension problem by delaying retirement. Such schemes are based on the proposition that improved health will make it possible for more people to work past age 65. However, the recent findings on the secular improvement in health at older ages make it clear that worsening health is not the explanation for the steep decline since 1890 in labor force participation rates of males over 65. As Costa (1993) has reported, the U.S. decline in participation rates of the elderly over the past century is largely explained by the secular rise in income and a decline in the income elasticity of the demand for retirement. It is also related to the vast increase in the supply and the quality of leisure-time activities for the laboring classes.

In Malthus's time, and down to the opening of this century, leisure was in very short supply in the OECD countries and, as Veblen pointed out (1934), it was conspicuously consumed by a small upper class. The typical person labored over 60 hours per week for wages and many had chores at home which consumed an additional 10 or 12 hours (Kuznets 1952; Fogel 1993a; Olson 1992; cf. Atack and Bateman 1992). Aside from sleep, eating, and hygiene, such workers usually had barely 2 hours a day for leisure. Although opera, theater, and ballet were available, they were too expensive to be consumed ordinarily by the laboring classes.

Over the twentieth century, hours of work have fallen by nearly half for typical workers. Ironically, those in the top decile of the income distribution have not shared much in this gain of leisure since the highly paid professionals and businessmen who populate the top decile work closer to the nineteenth-century standard of 3,200 hours per year than the current working-class standard of about 1,800 hours. There has also been a vast increase in the supply of leisure-time activities--movies, radio, television, amusement parks, participant and spectator sports, travel--

and a decline in the relative price of such activities. Many firms cater especially to the tastes of the elderly, offering reduced prices and special opportunities. As a result, the typical worker spends two-thirds as much time in leisure activities as in work and looks forward to retirement (Fogel 1992a and 1993a).

Given the growing and income-inelastic demand for leisure that characterizes the post-Malthusian milieu of the OECD nations, it remains to be seen to what extent the demand for leisure and retirement can be throttled. Policymakers may encounter as much resistance to efforts to reduce the implicit subsidies for leisure as they have had recently in raising the taxes on work.

### *6. Some Implications for the Theory and Measurement of Economic Growth*

Recent findings in the biomedical area call attention to what may be called the thermodynamic and physiological factors in economic growth. Although largely neglected by theorists of both the "old" and the "new" growth economics, these factors can easily be incorporated into standard growth models. Viewed in the human capital context, both factors may be thought of as labor enhancing technological changes that were brought about by developments in the agricultural, public health, medical services, and household sectors. They may also be thought of as adjustments for the mismeasurement of the labor input, when labor is measured only in person-hours.

I referred to the thermodynamic factor indirectly in section 5.1, when I indicated that about 30 percent of the British growth rate over the past 200 years was attributable to improvements in gross nutrition. That computation was based on the first law of thermodynamics, which holds that energy output cannot exceed energy input. Since that law applies as much to human engines as to mechanical ones, it is possible to use energy cost accounting techniques to estimate the increase in the energy available for work over the past two centuries. In the British case that increase had two effects. It raised the labor force participation rate by bringing into the labor force the bottom 20 percent of consuming units in

1790 who had, on average, only enough energy for a few hours of strolling. Moreover, for those in the labor force, the intensity of work per hour has increased because the number of calories available for work increased. This change in the intensity of effort, by itself, appears to have accounted for about 20 percent of the long-term growth rate.

The contention that the British intensity of effort increased over time may seem dubious since the work day, week, and year (measured in hours) declined significantly over the past two centuries. However, the British (and other Europeans) could not have worked at the same average intensity *per hour* in 1790 as they do today, since that would have required a considerably larger supply of dietary energy per capita than was actually available. Increases in the intensity of labor *per hour* was also a factor in the American case, where food supplies were far more abundant than in Europe. Even if it is assumed that the daily number of calories available for work was the same in the U.S. in 1860 as today, the intensity of work per hour would have been well below today's levels, since the average number of hours worked in 1860 was about 1.75 times as great as today. During the mid nineteenth century only slaves on southern gang-system plantations appear to have worked at levels of intensity per hour approaching current standards (cf. Fogel 1991 and 1993a; Olson 1992; Fogel and Engerman 1992).

The physiological factor pertains to the efficiency with which the human engine converts energy input into work output. Nutritionists, physiologists, and development economists have contributed to the extensive literature on this topic. Since some important issues are still unresolved, a firm assessment of the physiological contribution to economic growth is not yet possible. However, some aspects of the contribution can be indicated.

Changes in health, in the composition of diet, and in clothing and shelter can significantly affect the efficiency with which ingested energy is converted into work output.[7] Reductions in the incidence of infectious diseases increase the proportion of ingested energy that is available

for work both because of savings in the energy required to mobilize the immune system and because the capacity of the gut to absorb nutrients is improved, especially as a consequence of a reduction in diarrheal diseases. Thermodynamic efficiency has also increased because of changes in the composition of the diet, including the shift from grains and other foods with high fiber content to sugar and meats. These dietary changes raised the proportion of ingested energy that can be metabolized (increased the average value of the "Atwater Factors," to use the language of nutritionists). Improvements in clothing and shelter have also increased thermodynamic efficiency by reducing the amount of energy lost through radiation.

Individuals who are stunted but otherwise healthy at maturity will be at an increased risk of incurring chronic diseases and of dying prematurely. To evaluate the significance of changes in the rate of deterioration in the capacity to work over the life cycle one needs to calculate the effect of changes in stature and weight on the discounted present value of the difference between earnings and maintenance over the life-cycle (cf. Dasgupta 1993). A rare data set containing the desired information has been analyzed by R.A. Margo and R.H. Steckel (1982). The equation that they fitted to these data can be used to estimate the increase in the discounted net revenue stream at age 30 as a result of increasing the mean height and BMI from the levels that prevailed among the British in c.1790 to those of c.1980.[8] The exercise reveals that the discounted revenues would have increased by about 43 percent. This last figure, combined with a guess on the effect of the shifting of Atwater factors, suggests that the average efficiency of the human engine in Britain increased by about 60 percent between 1790 and 1980. The combined effect of the increase in dietary energy available for work, and of the increased human efficiency in transforming dietary energy into work output, appears to account for about 50 percent of the British economic growth since 1790.[9]

Focusing on the thermodynamic and physiological aspects of economic growth calls attention to the long lags that frequently occur between the time that certain investments are

made and the time that their benefits occur. Much of the gain in thermodynamic efficiency that occurred in Britain and other OECD countries between 1910 and 1980 was due to a series of investments made as much as a century earlier. Failure to take account of these extremely long lags between investments and payoffs leads to puzzling paradoxes. During the Depression Decade of the 1930s, for example, the U.S. unemployment rate was never less than 16 percent; for half the period unemployment ranged between 20 and 25 percent. Yet life expectation between 1929 and 1939 increased by 4 years and the heights of men reaching maturity during this period increased by 1.6 cm (U.S. Bureau of Statistics 1975; Karpinos 1958).

The resolution of the paradox turns, I believe, on the huge social investments made between 1870 and 1930 and whose payoffs were not counted as part of national income during the 1920s and 1930s even though they produced a large stream of benefits during these decades. I refer, of course, to the social investment in biomedical research (which included the establishment and expansion of modern teaching and research hospitals) whose largest payoffs came well after the investment was made. Also included in this category are such public health investments as the construction of facilities to improve the supply of water, the cleaning up of the milk supply, the draining of swamps, the development of effective systems of quarantines, and the cleaning up of the slums.

* * *

Keynes said: "In the long run we are all dead." That was an appropriate point to make during the interwar period, which included the severe inflations of the 1920s and the worst depression in history during the 1930s. Urgent, forceful action was needed to regain control of the money supply, to take care of the millions of unemployed, and to prevent the collapse of the democracies.

We live in another era. The major issues of economic policy in OECD nations today cannot be understood from a purely short-run perspective. The crisis in medical care, the

pension crisis, and the challenges of globalization are governed by long-run processes that policymakers need to understand. As I have tried to point out in this lecture, we have not yet completed the escape from hunger and premature death that began nearly three centuries ago. Chronic diseases and death are still occurring prematurely even in the rich countries. If the reforms of health care and pension programs now being considered by policymakers are to be successful, they must be consistent with the long-term physiological changes governing the decline in chronic diseases and the increase in longevity. Long-term forecasts that do not take account of the dynamics of these changes over the past century, and of the socioeconomic, biomedical, and other environmental improvements that made them possible, are liable to be far off the mark.

At the outset of this lecture I stressed the need for economists to take account of long-run dynamic processes through a study of history. Uncovering what actually happened in the past requires an enormous investment in time and effort. Fortunately for theorists, that burden is borne primarily by economic historians. Theorists only need to spend the time necessary to comprehend what the historians have discovered. A superficial knowledge of the work of economic historians is at least as dangerous as a superficial knowledge of theory.

## NOTES

1. Before proceeding with the discussion of chronic malnutrition it is necessary to clarify a terminological confusion that has misled some investigators: That is the distinction between the term "diet" or food intake (which represents gross nutrition) and the term "malnutrition" (which represents net nutrition--the nutrients available to sustain cellular growth). I will not dwell on this distinction here but will only emphasize that when I mean gross nutrition I will use the term *"diet"* and that such other terms as *"malnutrition," "undernutrition," "net nutrition,"* and *"nutritional status"* are meant to designate the balance between the nutrient intake (diet) and the claims on that intake. See Fogel and Floud (1994) for a further elaboration of this distinction.

2. See the appendix to Floud and Fogel 1994 for the estimated distributions of height, weight and the body mass index (BMI) in France c.1790.

3. In principle it is possible to construct size distributions of calories from household consumption surveys. Inasmuch as most of these surveys during the nineteenth century were focused on the lower classes, in order to make use of them it is necessary to know from what centiles of either the national caloric or the national income distribution the surveyed households were drawn.

4. Moreover, with a given population and technology, changes in the allocation of labor between agriculture and other sectors may lead to changes in body size and mortality. In an ancien régime economy the lower the share of the labor force that is in agriculture, cet. par., the lower the share of caloric production that can be devoted to baseline maintenance. The reasoning behind this statement is as follows: Assume that one worker in agriculture feeds himself plus three persons outside of agriculture. Hence, a movement of one percent of agricultural workers to nonagriculture would reduce the per

capita availability of food to the increased nonagricultural sector by about 1.33 percent. If baseline maintenance accounts for 75 percent of caloric consumption and if calories reserved for work remain constant in the nonagricultural sector, calories available for baseline maintenance in that sector would decline by about 1.8 percent (assuming that within the agricultural sector per capita production and consumption is unchanged).

5. Such possible synergies call into question the proposition that, because an individual appears to be currently well fed, malnutrition does not affect the outcome of diseases such as influenza, smallpox or typhoid (cf. Duncan, Scott, and Duncan 1993). Individuals may be more likely to succumb to such infections, even if they are currently well fed, because past malnutrition, either in utero or subsequently, has degraded vital organs (cf. Scrimshaw, Taylor, and Gordon 1968).

6. Premature mortality is defined as death rates that are higher than the 1980 death rates when standardized for the 1700 age structure (cf. Fogel 1986 and 1992b). Estimates of the overall death rate c.1790 are from Wrigley and Schofield (1981). Estimates of the relative death rates by deciles of the caloric consumption distribution were based on the estimated average heights and BMI in these deciles and on the relative mortality risks that they imply, as indicated in Figure 4. For further details on the computations see the appendix in Fogel and Floud 1994 and Table A2 in Fogel 1993b.

7. The discussion in this paragraph draws on Dasgupta 1993 and the sources cited there.

8. The data pertain to slaves seized as booty of war by the Union Army in 1863. The equation is:

$$\ln V = \underset{}{2.73} + \underset{(1.47)}{0.032S} + \underset{(0.92)}{0.17A} - \underset{(2.22)}{0.005A^2} + \underset{(-2.23)}{0.000046A^3} + \underset{(2.10)}{0.053H} - \underset{(2.16)}{0.019H} - \underset{(1.79)}{0.00027H \cdot W}; \quad \underset{(-1.73)}{}$$

$$N = 523; \; R^2 = 0.20;$$

where V is the value of a slave, S is a dummy for skin color, A is age, H is height (in

inches), and W is weight (in lbs). T- statistics are in parens. For 1790 I used 1.679m and 60.61 kg. For 1980 I used 1.76m and 75.89 kg.

9. The following procedure was used to arrive at this estimate: Bringing the bottom 20 percent of the caloric distribution of c.1790 into the labor force increased the labor force participation rate by 25 percent. Among those in the labor force the average number of calories available for work increased by 56 percent between c.1790 and 1980. Hence, the total increase in output per capita as a result of the increased availability of calories for work was 95 percent (1.25 x 1.56 = 1.95). Dasgupta's (1993) discussion suggests that reductions in diarrheal and other diseases combined with a shift in the compositon of the diet increased the Atwater factors by about 12 percent. Since the exercise based on the equation in note 8 implies that the reduction in chronic diseases and premature mortality increased thermodynamic efficiency by 43 percent, the combined increase in thermodynamic efficiency is about 60 percent (1.12 x 1.43 = 1.60). In combination, then, increased calories available for work and the increased thermodynamic efficiency increased per capita income between c.1790 and 1980 by 212 percent (1.95 x 1.60 = 3.12) or by 0.60 percent per annum ($3.12^{(1/190)} - 1 = 0.0060$), which is slightly more than half of the annual British growth rate (0.60 ÷ 1.15 = 0.52). For further details, including discussion of possible upward and downward biases in this computation, see Fogel 1987 and Fogel and Floud 1994.

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Table 1
Estimated Average Final Heights of Men Who Reached Maturity
Between 1750 and 1875 in Six European Populations,
by Quarter Centuries
(cm)

| | (1) Date of maturity by century and quarter | (2) Great Britain | (3) Norway | (4) Sweden | (5) France | (6) Denmark | (7) Hungary |
|---|---|---|---|---|---|---|---|
| 1. | 18-III | 165.9 | 163.9 | 168.1 | – | – | 168.7 |
| 2. | 18-IV | 167.9 | – | 166.7 | 163.0 | 165.7 | 165.8 |
| 3. | 19-I | 168.0 | – | 166.7 | 164.3 | 165.4 | 163.9 |
| 4. | 19-II | 171.6 | – | 168.0 | 165.2 | 166.8 | 164.2 |
| 5. | 19-III | 169.3 | 168.6 | 169.5 | 165.6 | 165.3 | – |
| 6. | 20-III | 175.0 | 178.3 | 177.6 | 172.0 | 176.0 | 170.9 |

*Sources:* Fogel 1987, Table 7 for all columns except 5. Col. 5: Rows 3-5 were computed from Meerton 1989 as amended by Weir (1992), with 0.9 cm added to allow for additional growth between age 20 and maturity (Gould 1869, 104-105; cf. Friedman 1982, 510, n. 14). The entry to row 2 is derived from a linear extrapolation of Meerton's data for 1815-1836 back to 1788, with 0.9 cm added for additional growth between age 20 and maturity. The entry in row 6 is from Fogel 1987, Table 7.

Table 2
A Comparison of the Probable French and English Distributions
of the Daily Consumption of Kcals per Consuming Unit
Toward the End of the Eighteenth Century

| | A | B |
|---|---|---|
| | France c. 1785 | England c. 1790 |
| | $\bar{X}$ = 2,290 | $\bar{X}$ = 2,700 |
| | $(s/\bar{X})$ = 0.3 | $(s/\bar{X})$ = 0.3 |

| | Decile (1) | Daily kcal consumption (2) | Cumulative % (3) | Daily kcal consumption (4) | Cumulative % (5) |
|---|---|---|---|---|---|
| 1. | Highest | 3,672 | 100 | 4,329 | 100 |
| 2. | Ninth | 2,981 | 84 | 3,514 | 84 |
| 3. | Eighth | 2,676 | 71 | 3,155 | 71 |
| 4. | Seventh | 2,457 | 59 | 2,897 | 59 |
| 5. | Sixth | 2,276 | 48 | 2,684 | 48 |
| 6. | Fifth | 2,114 | 38 | 2,492 | 38 |
| 7. | Fourth | 1,958 | 29 | 2,309 | 29 |
| 8. | Third | 1,798 | 21 | 2,120 | 21 |
| 9. | Second | 1,614 | 13 | 1,903 | 13 |
| 10. | First | 1,310 | 6 | 1,545 | 6 |

Sources and procedures: See Fogel 1987, esp. tables 4 and 5 and note 6.

**Table 3**

**Comparison of the Prevalence of Chronic Conditions Among Union Army Veterans in 1910, Veterans in 1983 (Reporting whether they ever had specific chronic conditions), and Veterans in NHIS 1985-88 (Reporting whether they had specific chronic conditions during the preceding 12 months), Aged 65 and Above, Percentages**

| Disorder | 1910 Union Army veterans[a] | 1983 veterans[a] | Age-adjusted 1983 veterans[a] | NHIS 1985–88 veterans[a] |
|---|---|---|---|---|
| Musculoskeletal | 67.7 | 47.9 | 47.2 | 42.5 |
| Digestive | 84.0 | 49.0 | 48.9 | 18.0 |
| Hernia | 34.5 | 27.3 | 26.7 | 6.6 |
| Diarrhea | 31.9 | 3.7 | 4.2 | 1.4 |
| Genito-urinary | 27.3 | 36.3 | 32.3 | 8.9 |
| Central nervous,endocrine, Metabolic, or blood | 24.2 | 29.9 | 29.1 | 12.6 |
| Circulatory[b] | 90.1 | 42.9 | 39.9 | 40.0 |
| Heart | 76.0 | 38.5 | 39.9 | 26.6 |
| Varicose veins | 38.5 | 8.7 | 8.3 | 5.3 |
| Hemorroids[c] | 44.4 | | | 7.2 |
| Respiratory | 42.2 | 29.8 | 28.1 | 26.5 |

[a] Prevailing rates of Union Army veterans are based on examinations by physicians. Those for the 1980s are based on self reporting. Comparison of the NHIS rates with those obtained from physicians' examinations in NHANES II indicates that use of self reported health conditions does not introduce a significant bias into the comparison. See the source for a more detailed discussion of possible biases and their magnitudes.

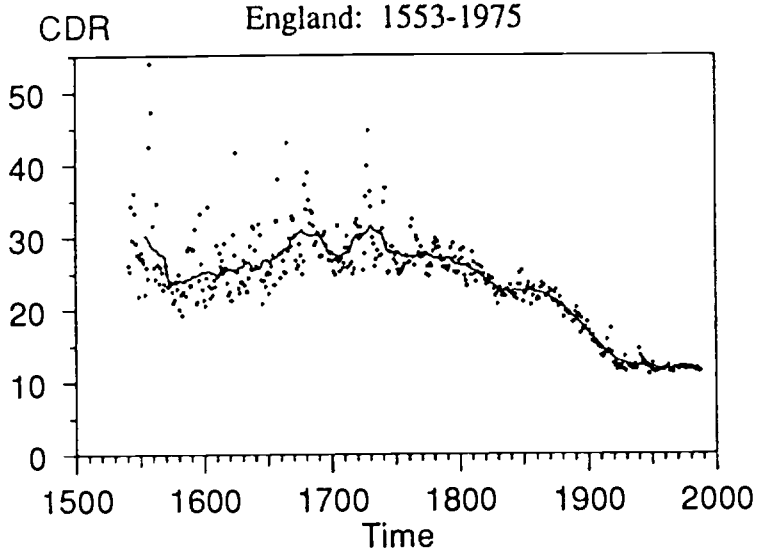
[b] Among veterans in 1983, the prevalence of all types of circulatory diseases will be underestimated because of under-reporting of hemorroids.

[c] The variable indictating if the 1983 veteran ever had hemorroids is unreliable.

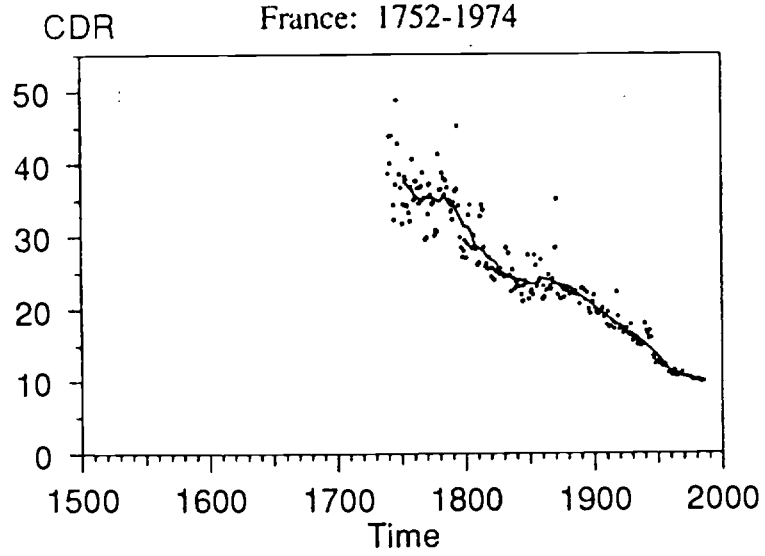
*Source:* Fogel, Costa, and Kim 1993

**FIGURE 1**
The Secular Trends in Mortality Rates
in England and France

PART A
England: 1553-1975




PART B
France: 1752-1974



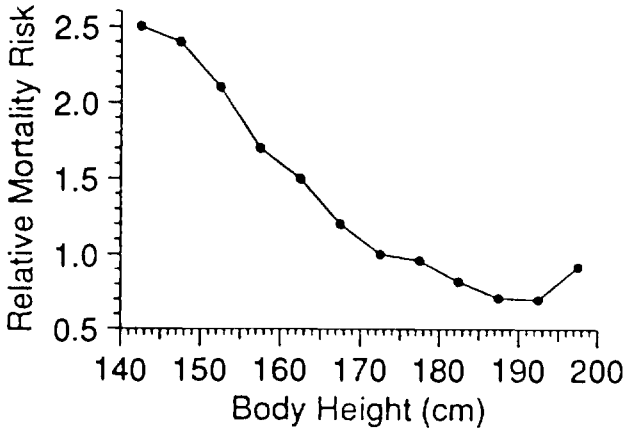

*Note:* Each diagram shows the scatter of annual death rates around a 25-year moving average. See Fogel and Floud 1991 for sources and procedures.

**FIGURE 2**

Comparison of the Relationship between Body Height and Relative Risk in Two Populations

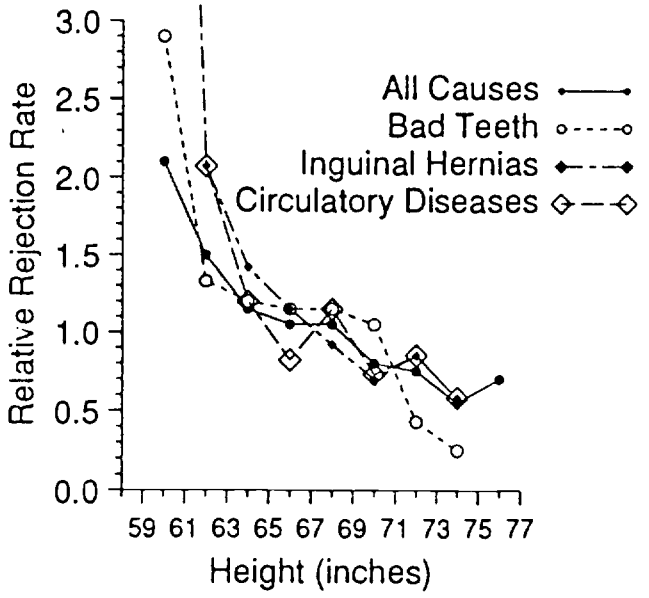
PART A

Relative Mortality Risk among Norwegian Men Aged 40-59, between 1963 and 1979




PART B

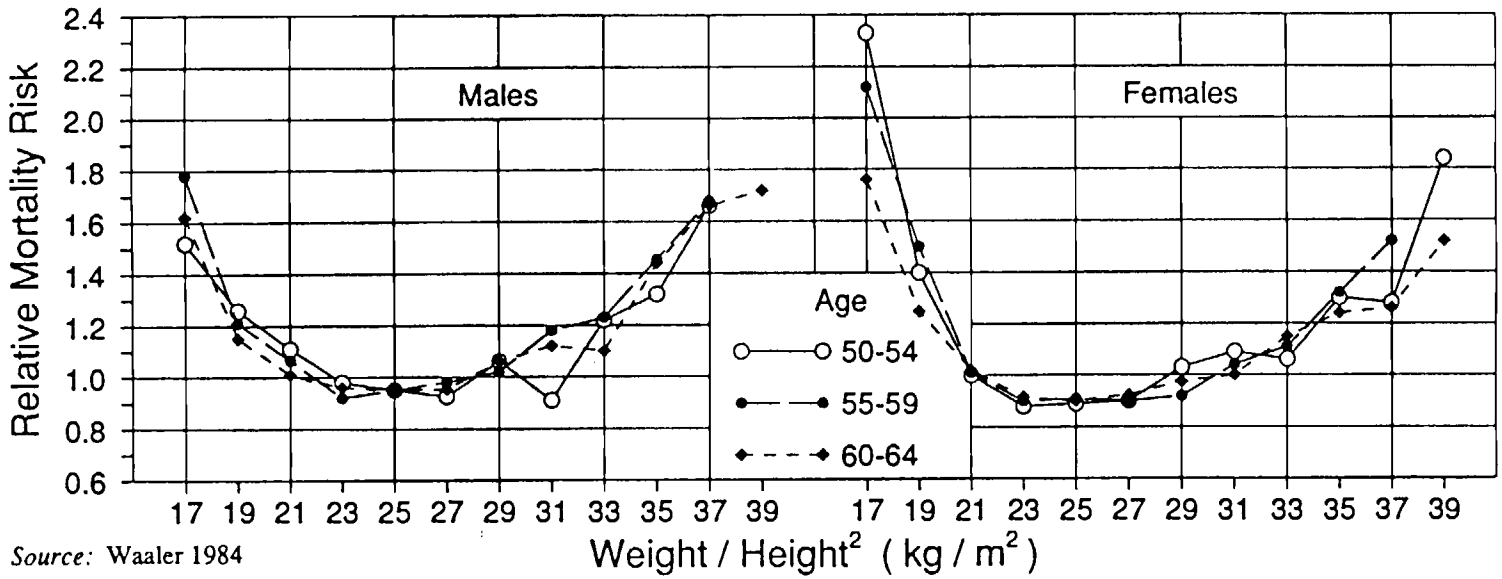
Relative Rejection Rates for Chronic Conditions in a Sample of 4,245 Men Aged 23-49, Examined for the Union Army




*Sources:* Part A: Waaler 1984, Part B: Fogel 1993b

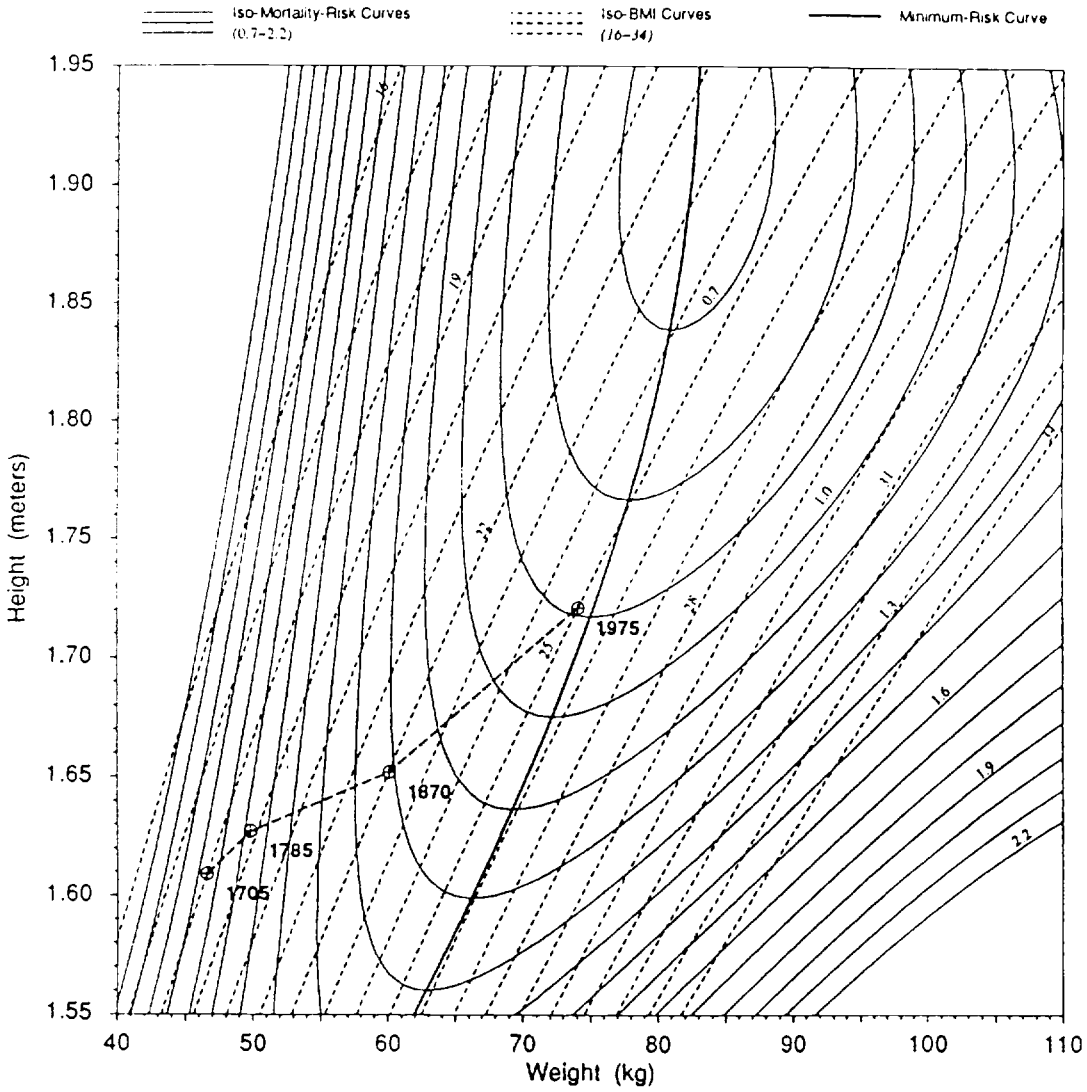
**FIGURE 3**

Relationship between BMI and Prospective Risk among Norwegian Adults Aged 50-64 at Risk (1963-1979)



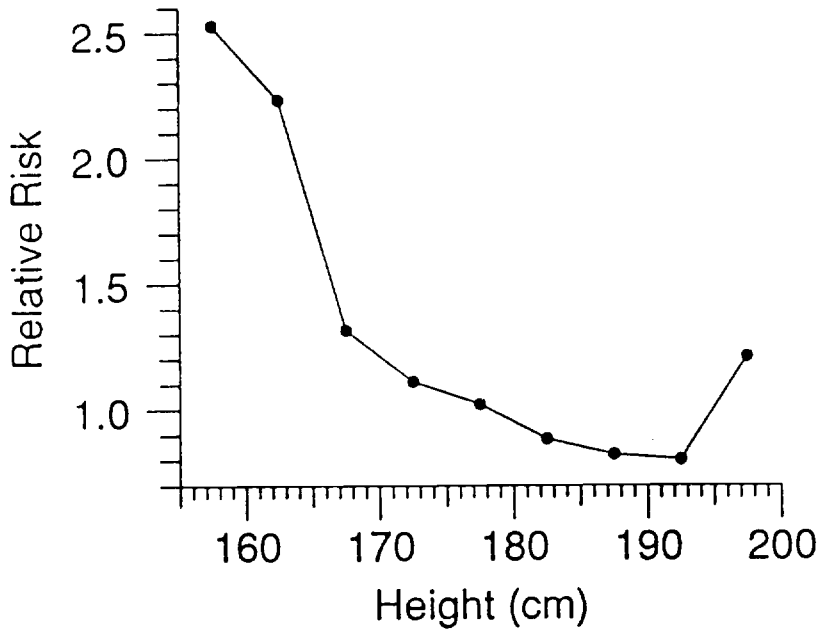

*Source:* Waaler 1984

**FIGURE 4**
**Iso-Mortality Curves of Relative Risk for Height and Weight Among Norwegian Males Aged 50-64, With a Plot of the Estimated French Height and Weight at Four Dates**



## FIGURE 5

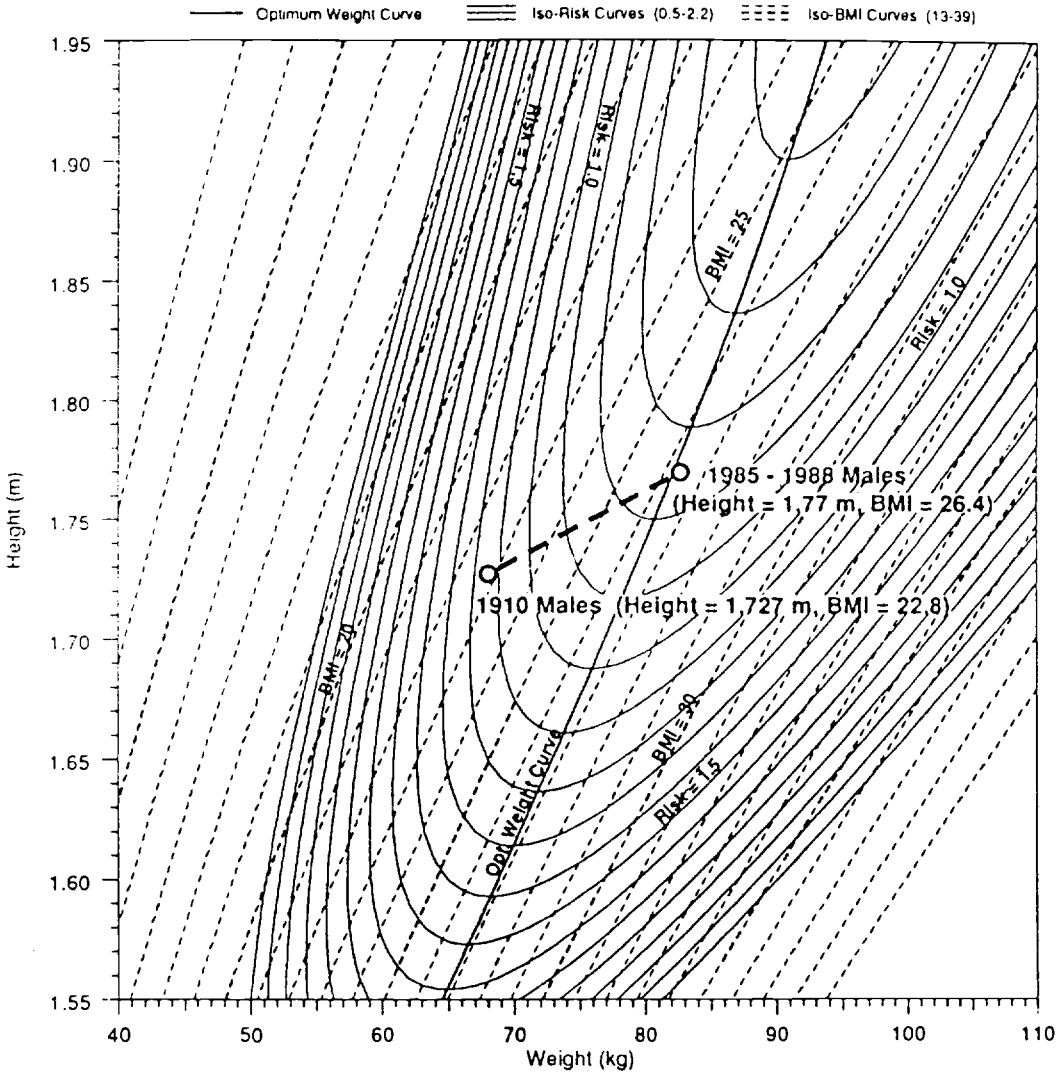
The Relationship between Height and Relative Risk of Ill Health in NHIS Veterans Aged 40-59




*Source:* Fogel, Costa, and Kim 1993

**FIGURE 6**

Health Improvement Predicted by NHIS 1985-1988 Health Surface

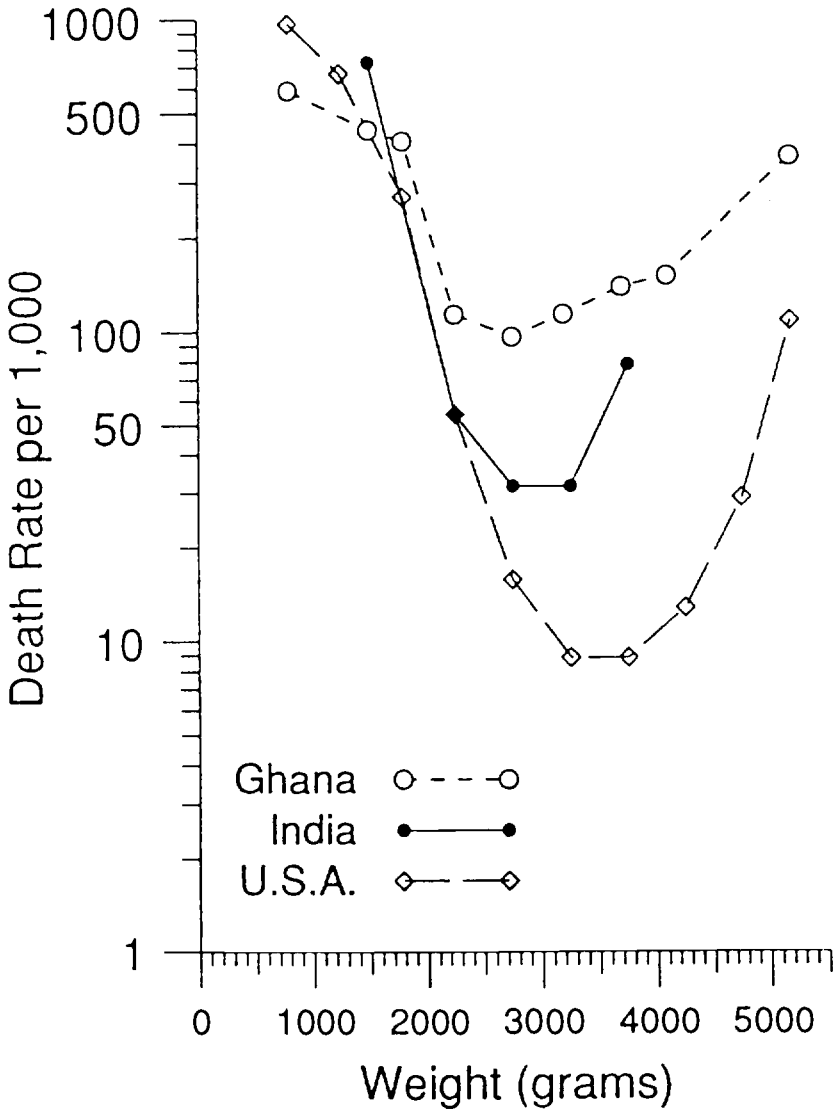



All risks are measured relative to the average risk of morbidity (calculated over all heights and weights) among NHIS 1985-1988 white males aged 45-64.

*Source:* Kim 1993

## FIGURE 7

Perinatal Death Rate by Birth Weight
in Ghana, India, and U.S.A.




*Source:* Hytten and Leitch 1971